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THE PATHOGENIC EFFECT OF ELECTRICAL CURRENT

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16. Abstract

The history of study of injuries due to electric current is traced. The effect of physical parameters (current path, voltage, resistance, current strength, etc.) of the condition of the organism, and environmental factors on electrotrauma is investigated. Forms of death attributable to the effects of electric current are described. Treatment is devoted to electric shock and exogenic effects, and prophylaxis and therapy of electrotraumata are briefly considered.

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THE PATHOGENIC EFFECT OF ELECTRICAL CURRENT

G. L. Frenkl'[1], K. A. Azhibayev[2] and I. K. Mishchenko[3]

The first systematic investigations on electrotrauma were begun at the end of the 19th century and the beginning of the 20th century (Prevost, Batelli, 1899, 1900: Jellinek, 1932; Freiberger, 1934; Ferris, King, Spenca, Williams, 1936). In the Soviet Union, I. R. Petrov should be considered the founder of electropathology. Over the period 1930-1950, together with his associates, I. R. Petrov conducted a large number of investigations devoted to this problem. N. A. Vigdorshik (1940) summarized the entire world-wide literature on electropathology in a monograph which maintains its importance up to the present time. /145*

After the end of World War II, investigations in the study of electropathology were intensively conducted in the Kirghiz SSR under the supervision of G. A. Frenkel'. The basic results of the investigations conducted by the Kirghiz group of electropathologists were published in the materials of conferences on electrotrauma — the Republican Conference (1957) and the All--Union Conference (1962), and in thematic collections, as well as in the dissertations of K. A. Azhibayev (1955), A. S. Sultanaliyev (1957), V. Ya. Eskin (1962), I. K. Mishchenko (1963), A. B. Baybosunov (1967), A. Kh. Karaseva (1967) and others.

Intensive work in studying the pathogenesis and therapy of injuries caused by electrical current was conducted in the laboratory of V. A. Negovskiy.

Besides pathophysiologists, engineers (V. Ye. Manoylov, A. P. Kiselev, G. S. Solodovnikov, etc.) have been occupied with a study of problems of electropathology.

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*Numbers in the margin indicate pagination in the foreign text.

The biological effect of electrical current is determined by its physical parameters, and also by the condition of the organism. Here, we shall not use the term widespread in the electropathological literature, "sensitivity" of the organism to electrical current, but rather we shall use the reverse, but more correct, concept of "resistance".

The characteristic peculiarity of electrical current, as an extreme stimulus, is its pronounced "biirradiation" (K. A. Azhibayev, D. A. Alymkulov and B. M. Shapiro, 1962)[4].

The animal organism is a "third class conductor" (V. Ye. Manoylov, 1961), inasmuch as properties of first and second class conductors are manifested here in a form altered by biological principles. /146

The effect of an electrical current on the organism is manifested in the electrical, thermal and mechanical effect (S. Yellinek, 1927; I. R. Petrov, 1947; N. A. Vigdorchik, 1940; G. L. Frenkel', 1945; Fischer and Frolicher, 1951). However, of greatest interest for electropathology is the so-called specific effect of current caused by redistribution of ions (Nernst, 1908; P. P. Lazarev, 1945). In the foreign literature, occasionally the specific effect of a sinusoidal current, on the strength of its variable polarity, is called "vibrator current".

The effect of an electrical current on the organism frequently leads to the development of extremal conditions accompanied by sharp disorders in the cardiac activity (cardiac fibrillation), to sharp disorders in respiration, and to the development of shock reactions.

The Effect of Physical Parameters of the Electrical Current on Electrotrauma

The Effect of the Current Path. Standard variants of paths of a current passage in the body are shown in Figure 32. In experiment, current is usually passed along these "loops" to obtain electrotrauma. The loops indicated by

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[4]"Biirradiation" — the effect from the action of an electrical current on the organism. It is additive, built up from the direct effect of electrical current on cells, tissues, and organs, and indirectly as the result of current irritation of the extero- and interoreceptors lying on its path. "Biiradiation" is not a characteristic unique to electrical current.

numbers 1, 2, 3, 4 and 5 ensure passage of the current through the heart and are therefore most dangerous for man and the dog. The lower (in animals — hindmost) loop is unique (see Figure 32, 9). When a current is passed along the lower loop of an animal as sensitive to it as a dog, cardiac fibrillation is not successfully attained even at high voltage.

W location	1	2	3	4	5	6	7	8	9	10
Diagram of current path										
Current designation	J_1	J_2	J_3	J_4	J_5	J_6	J_7	J_8	J_9	J_{10}
Resistance designation	r_1	r_2	r_3	r_4	r_5	r_6	r_7	r_8	r_9	r_{10}
Name of loop	Full	Right full	Left full	Right oblique	Left oblique	Right	Left	Upper	Lower	Transverse

Figure 32. Standard "Loops" (N. A. Vigdorchik, 1940; G. L. Frenkel', 1944).

The Significance of Voltage and Resistance. Electrotrauma is conventionally divided into the following classifications: low voltage, when voltage does not exceed 1,000 V, and high voltage, when voltage exceeds 1,000 V. Fischer and Froelicher (1951) also introduce the concept of "super high voltage electrotrauma" (voltage of hundreds of kv, injury by lightning).

In injuries by current of different voltages, the degree of pronouncement of the responsive reaction varies; data in the literature indicate that in the range of 200-800 V there is little change with an increase in voltage (Wegelin, 1935; I. R. Petrov, 1936). K. A. Azhibayev (1964), reached other conclusions /147
which show that with an increase in voltage even in this interval, the danger of the current increases. It is entirely possible that this difference arose as the result of the fact that K. A. Azhibayev worked under the special climato-meteorological conditions of Central Asia.

So long as the epidermis is intact, a gradual increase in voltage leads to a less pronounced increase in current. But as soon as a "histological" breakthrough occurs, the current increases stepwise and subsequently the body of the animal organism acts as a purely ohmic resistance. According to the data of various authors, the magnitude of breakthrough voltage varies greatly.

Thus, V. I. Korol'kova (1956), indicates a range of 10-15 V, P. I. Podol'skiy (1946), considers that breakthrough can also occur at lower voltages, while A. F. Pakhomov (1959) determined this magnitude to be 43 V. The electrical resistance of human skin, according to V. Ye. Manoylov (1966) is within the limits of 280-400 V/mm.

The reaction of the organism to the effect of electrical current primarily depends upon the magnitude of the invasive current and its form. The magnitude of the invasive current (old designation — "current strength") is linked to its voltage and to the body resistance of the affected organism by the known formula:

$$I = \frac{V}{R},$$

which expresses Ohm's law. Here I is expressed in amperes, V in volts and R in ohms. In this form the formula is used in a case in which the resistance is active (purely ohmic). If the circuit also contains capacitances, and the resistances are inductive (they comprise the reactive component and are occasionally called "apparent"), then total (field) resistance (active plus reactive) of the circuit is called impedance and is designated by Z. In this case, Ohm's formula acquires the following form:

$$I = \frac{V}{Z}.$$

Inasmuch as there are no metal conductors in biological systems (i.e., no condition for the appearance of inductive resistances), impedance only includes the type of reactive resistance which is caused by the presence of capacitances.

Various replacement systems for one cell (D. L. Rubinshteyn, 1947) and for a whole organism (V. Ye. Manoylov, 1966; A. F. Pakhomov, 1961, and others) have been suggested. In the capacity of an equivalent system for a whole organism, the system of A. F. Pakhomov (Figure 33) is given. In it R_s is the active resistance of the skin; R_i active resistance of the internal organs; C capacitance of the skin between the surface of the electrode and the subcutaneous tissue.

Inasmuch as the resistance of the body of an animal depends upon voltage and frequency of the current and upon its "loop", and while different authors have made their measurements under various conditions, so in the literature there is a vast scatter of values of resistance — from 1,000 to 1,000,000 ohms. But this pertains only to total resistance, the basic fraction of which is determined by the keratic layer of the epidermis. With respect to internal resistance (R_i in Figure 33), in this regard the authors are quite agreed and estimated it at 700-1,000 ohms (I. P. Tishkov, 1886; Loebl, 1932; I. P. Petrov, 1947; K. A. Azhibayev and I. K. Mishchenko, 1966). Important is the fact that /148 resistance changes as a function of the condition of the nervous system — for example, pain decreases resistance.

In this system, no provision is made for the so-called transition resistances, i.e., resistances between electrodes (or conductors) and the organism. In the experiment, they are made particularly small, combing wool and using separators soaked in electrolytes.

N. B. Poznanskaya (1938) showed that the resistance of the skin in various parts of the body is not identical. This macromosaicism has quite large areoles. But macromosaicism of the skin resistance also exists (Croon, 1953; V. Ye. Manoylov, 1966; Lullies, Rumberger, 1966).

The Significance of Current Strength. The magnitude of response reaction of the animal organism to the effect of electrical current primarily depends upon current strength. This factor will therefore be examined in greater detail here. Data of the literature which are available on this question primarily pertain to alternating current at 50-60 Hz.

Summary tables of current values which vary in their effectiveness have been published. N. A. Vigdorchik (1940), I. R. Petrov (1947), Koeppen and Pance (1955) divided currents having different values into 4 ranges. The Koeppen and Pance (1955) classification is given below.

The first range is made up from 0.5 to 2.0 to 25 mA. The threshold value of an irritating current is within the limits of 0.5-2.0 mA. An increase in current strength beyond the limits of threshold of sensation is accompanied by an increase in muscular reaction; then convulsions appear in the extremities, and upon reaching a current strength of 15-25 mA convulsions become so powerful that a man is not capable of independently breaking contact with the electrodes ("unreleasable current", after I. R. Petrov).

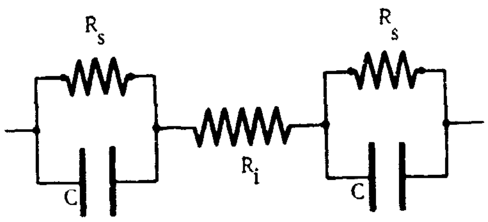

Figure 33. Equivalent System of Whole Organism Resistance (A. F. Pakhomov, 1961).

The second range includes currents from 25 to 80 mA. "Unbreakable current", during short term exposure, does not pose any direct danger for lives. With more prolonged "contact" with the current--carrying object, currents of this magnitude can lead to death, causing convulsions of the respiratory muscles, and as a result of this — acute electrical asphyxiation.

Currents capable of causing cardiac fibrillation belong to the third range. These currents are within limits of from 80 mA to 3 A.

According to data obtained by Ferris, King, Spens and Williams (1936), in experiments performed on dogs, sheep, pigs and calves, the minimum current magnitude causing cardiac fibrillation is directly related to body weight and heart weight of these animals. These authors concluded that for man a current in excess of 100 mA must be considered lethal.

It has been established that the danger of appearance of cardiac fibrillation during exposure of an organism to alternating current is determined by the magnitude of the current flowing directly through the heart.

With an increase in the strength of the current flowing through the heart beyond the limits of its minimum value causing fibrillation, the current gradually loses this capacity. Such currents belong to the fourth range. Thus, /149
with placement of the electrodes on the exposed heart of the cat, cardiac fibrillation appeared at a current strength of 2.5-7.5 mA and was absent with an increase in current strength to 300 mA (L. S. Poz, 1952). With application of the electrodes to the surface of the thoracic cavity, the minimum current value not causing fibrillation (minimum defibrillating current) is 1.5-2 A for a dog weighing 5-8 kg, and 6 A for dogs weighing 13 kg (N. L. Gurvich, 1957). In experiments performed on sheep, passing an alternating current (60 Hz) along the "right oblique loop" for a period of 0.03 seconds (in the "vulnerable phase" of the cardiac cycle), Ferris et al., (1936), showed that current strength of 1 A does not cause fib lation, shocks of a current of 4 A were

accompanied by fibrillation in most cases, shocks of a current of 12 A caused fibrillation only in 1/5 of the cases, while 24 A entirely lost the capacity to cause fibrillation but acquired the capacity to cause prolonged respiratory arrest.

Prevost and Battelli (1899) first showed in experiments performed on dogs and small animals that with an increase in voltage of the invasive current within certain limits, its fibrillating capacity decreases, and dependent upon the value of voltage there is predominant damage to the organs of respiration or circulation. According to their data, current having a voltage of 110 to 600 V, as a rule, caused death from primary cardiac arrest, while current having a voltage of 600 to 1,200 V caused death primarily as the result of simultaneous respiratory and cardiac arrest; current with a voltage in excess of 1,200 volts cause death as the result of primary respiratory arrest. This same principle was later confirmed by Wolter (1930), M. P. Brestkin, A. V. Lebedinskiy, L. A. Orbeli and V. V. Strel'tsov (1932), I. R. Petrov (1936) and others. These authors noted that high voltage current yields a smaller percentage of lethal injuries than lower voltage current, and that with high voltage it is primarily the respiratory apparatus which is injured. Since primary respiratory arrest is decreased by elementary methods, the outcome of high voltage electrotrauma is more favorable. This is also confirmed in the data of Kouwenhoven and Milnor, which is given in Table 15.

TABLE 15. COMPARATIVE RESULTS OF USING ARTIFICIAL RESPIRATION IN INJURIES CAUSED BY HIGH VOLTAGE AND LOW VOLTAGE CURRENT (KOUWENHOVEN, MILNOR, 1957)

Voltage, V	No. of injuries	No. of those revived by artificial respiration
2 000—80 000	68	18
110—480	12	1

The Time Factor. The duration of the current action has real significance for the organism's response reaction. Thus, long term contact with a current carrying object can become dangerous due to developing asphyxiation.

The significance of the time factor has been best studied with respect to the appearance of electrical cardiac fibrillation caused by alternating current (60 Hz). A decrease in the duration of action of current from 3 to 0.1 seconds and less leads to an increase nearly 10 times in the threshold fibrillation current.

Ferris et al., showed that increase in duration of current action from 3 to 12 seconds is accompanied by a decrease in the threshold of fibrillation. The same principle was established by I. K. Mishchenko (1962) in experiments on /150 dogs. An increase in the duration of current action only from 12 to 30 seconds led to a decrease in the threshold of fibrillation.

I. R. Petrov and V. E. Glazenat (1939), in examining the significance of the time factor, took as criteria of danger not the threshold of fibrillation, but rather a change in arterial pressure and respiration. Five stages of these disorders were arbitrarily isolated (Table 16).

TABLE 16. LINK BETWEEN DURATION OF CLOSING THE CIRCUIT OF ALTERNATING CURRENT AND RESPONSIVE REACTION OF THE ORGANISM (AFTER I. R. PETROV AND V. E. GLAZENAT)

Time, seconds	Current strength, mA					
	4,5–15	16–30	31–50	51–100	101–300	300
0,01–0,02	–	–	+–	+–	+	+
0,03	+–	+–	+–	+–	+	++
0,0[illegible]	+–	+–	+–	++	++	++
0,0[illegible]	+–	+	+	++	++	++
0,09	+–	+	++	++	++	++
0,15	+	++	++	++	++	+++
0,25	+	++	++	++	+++	+++
0,4–0,5	+	++	++	++	+++	+++
0,6–0,7	+	++	++		+++	+++
1	+	++	++	+++	+++	+++

Designations: – absence of change in respiration and arterial pressure; +– weakly pronounced changes in respiration and arterial pressure; + more pronounced changes in respiration and arterial pressure (together or individually); ++ clearly defined changes in arterial pressure; +++ severe reaction.
Note: Commas indicate decimal points.

<u>The Significance of the Class of Current</u>. Voltages of direct and alternating current, equivalent in their effect on the organism, are respectively, 120 and 42 V (A. S. Sultanaliyev, 1957). Kouwenhoven and Langworthy (1930), showed that direct current is less dangerous than alternating current only up to a voltage of 500 V. At a voltage of 500 V, the danger of both classes of current is equal, while in voltages of more than 500 V direct current is more dangerous.

These data were also later confirmed by V. Ye. Manoylov, A. F. Pakhomov and G. S. Solodovnikov (1953) on white mice, and by K. A. Azhibayev (1957) on dogs.

In work with direct current, the question of the significance of the direction of current unavoidably arises, i.e., is the current ascending (cathode on the cranial part of the body, anode on the caudal) or vice versa. Ascending direct current is more dangerous than descending current of the same voltage (Wilcke, Broghammer, 1956; K. A. Azhibayev, 1957).

Wilcke and Broghammer explain this by the fact that the cathode increases excitation, while the anode suppresses it. Therefore, with the ascending direction of the current the sinus ganglion of the heart is under the accelerating influence of the cathode, while the apex is under the suppressing effect of the anode. With a descending current, the sinus ganglion is suppressed by the anode, while the excitability of the apex is increased by the cathode. Stimulus proceeding from the sinus ganglion with the ascending current encounters ever increasing suppression of conductivity on its path. When this suppression of conductivity becomes lower than the critical value (12 cm/sec, after Trautwein, 1950), fibrillation ensues. With a descending current, the wave of stimulus, proceeding from the anode-suppressed sinus ganglion, is hastened by the cathode during its propagation. Therefore, with a descending current all conditions exist for the appearance of fibrillation over the course of the entire time the circuit is closed, while at the same time with a descending current these conditions exist only at the moment of circuit break.

Extremely demonstrative is the experiment where two dogs are simultaneously included in the circuit such that a direct descending current runs through one, while an ascending directed current runs through the other (K. A. Azhibayev). Here, the primary injurious parameter — current value is strictly equal. As one sees from Figure 34, the dog connected in the ascending direction dies, while at the same time the dog connected in the descending direction remains alive. In white mice, in which irreversible cardiac fibrillation does not appear, the direction of direct current with other conditions being equal has no significance (A. S. Sultanalivev, 1959).

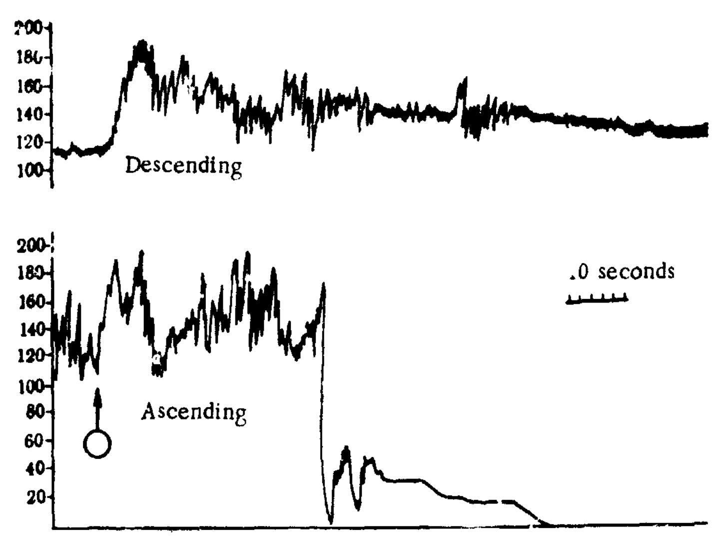

Figure 34. Simultaneous Successive Inclusion Into a Direct Current Circuit of Two Dogs: One on the Descending Direction (Upper Kymogram of Arterial Pressure), the Other on an Ascending Direction (Lower Curve of Kymogram). The arrow indicates the moment of closing the circuit (K. A. Azhibayev, 1957).

The Role of Frequency of Alternating Current. An investigation of the role of frequency of an electrical current was in its time the cause for much dispute as the result of the fact that different authors use different objects of observation and different physiological criteria. The most effective frequency for causing fibrillation in large animals is a current frequency within the limits of 40-60 Hz. The danger of current for these animals diminishes both with respect to increasing the frequency beyond the limits of 60 Hz (Wolter, 1930, 1934; S. F. Lidikh, I. A. Kanevskiy, 1935; Kouwenhoven, Hooker, Lotz, 1936: B. I. Kadykov, B. V. Orlov, 1958; K. A. Azhibayev and V. Ya. Eskin, 1960, and others), and with respect to decreasing the frequency below 40 Hz (Wolter, 1930, 1934; Kouwenhoven, Hooker and Lotz, 1936; Ferris et al., 1936).

Hence, all the investigators who studied the effect of alternating current /152 on the hearts of large animals concluded that the frequency of electrical current on an order of 50 Hz is most dangerous with respect to the appearance of fibrillation.

In spite of traditional viewpoints to the effect that the danger of alternating current decreases on both sides of the critical frequency (50 Hz), reports have appeared (Dalziel, 1943, 1953), that in a range of at least 50-1,000 Hz a decrease in the injurious effect of electrical current on man is not observed. G. S. Solodovnikov and O. D. Ushinskaya (1957), having affirmed the proposition of Dalziel, found, moreover, a coincidence of results with respect to the parameters of the motor reaction (in people and rabbits), and with respect to a lethal outcome (on white mice).

K. A. Aznibayev, I. K. Mishchenko, v. Ya. Eskin, M. T. Turkmenov, and G. L. Frenkel' (1964), showed that the frequency of 200 Hz is most dangerous with respect to the effect of respiratory arrest.

Experiments set up by K. A. Azhibayev and V. Ya. Eskin (1960), under conditions of decreased atmospheric pressure, particularly clearly revealed an increase in the danger of respiratory death with currents of increased frequency in the range of 50-200 Hz (Figure 35).

K. A. Azhibayev and his associates (1964) investigated the comparative effect of various current frequencies in their elementary mixture. In the capacity of criteria for estimating the effect of currents, fibrillation of the /153 heart and electrical asphyxiation were chosen. It turned out that during the action of two frequencies in the range of 50-10,000 Hz, varying in direction, the fibrillating action depends on the frequency of mixture components (Figure 36).

As is seen from this figure, leading significance for the fibrillating effect of the current belongs to the lowest frequency component of any mixture[5].

[5]The practical significance of investigating mixtures of several frequencies see in the book: I. K. Akhunbayev and G. L. Frenkel', 196 .

Proportional to its increase, the worsening effect of the second frequency decreases and with mixtures of 50 + 2,000 Hz, the significance of the second frequency actually disappears. We note that the fibrillating effect of mixtures of near frequencies, which differ from each other by 50 to 100 Hz, increases. This occurs as the result of the formation of beats having frequencies of 50-100 Hz, which are most dangerous for cardiac damage (the drop in curves a and b in Figure 36 is the so-called crutch phenomenon).

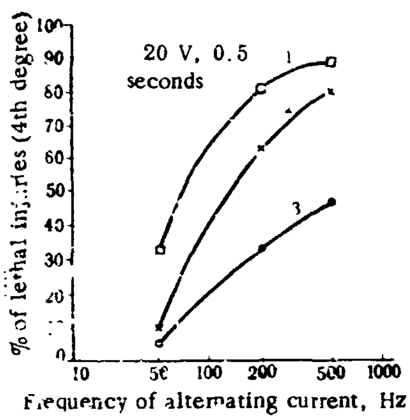

Figure 35. The Curve of the Dependence of Lethal Injuries on Frequency of Current and Height Above Sea Level (Experiments in the Pressure Chamber) (K. A. Azhibayev, V. Ya. Eskin, 1960). Height above sea level: 1, 4,000 m; 2, 2,000 m; 3, 760 m.

The Effect of the Condition of the Organism and Environmental Factors on Electrotrauma

The Significance of the Condition of the Organism. The biological effect of electrical current significantly depends on the original condition of the organism. S. Yellinek (1927) noted that electrotrauma received during sleep occurs more favorably. This is apparently related to inhibition of the cerebral cortex. F. M. Danovich (1947), in an experiment on mice, showed that ether anesthesia decreases the danger of acute electrotrauma by more than 15 times. Similar data have also been obtained on other species of animals (V. N. Proskurin and S. P. Petrenko, 1962; S. V. Yakushevich and G. M. Yevel'kin, 1962; V. N. Boytsov and Yu. M. Zimenko, 1962; M. S. Chekan and S. V. Afanas'yev, 1962). It is notable that in "hypnotic sleep", the danger of electrotrauma also decreases (V. N. Proskurin and S. P. Petrenko, 1962).

Alcohol drunkenness decreases resistance to electrical current (Manoilov, 1935; Ajello, 1935). Apparently, the degree of resistance to current depends upon the depth of drunkenness all the same (G. L. Frenkel', 1944): drunkenness up to the degree of narcosis acts as the latter. A certain significance in the mechanism of the injurious effect of electrical current attaches to the degree of oxygen starvation. Deep asphyxia decreases the resistance of the organism to electrical current (P. P. Goncharov, 1939; I. K. Mishchenko, 1963). However, K. A. Azhibayev and I. K. Mishchenko (1966), came to contradictory conclusions. This difference is apparently related to the different degree of asphyxia which these authors caused.

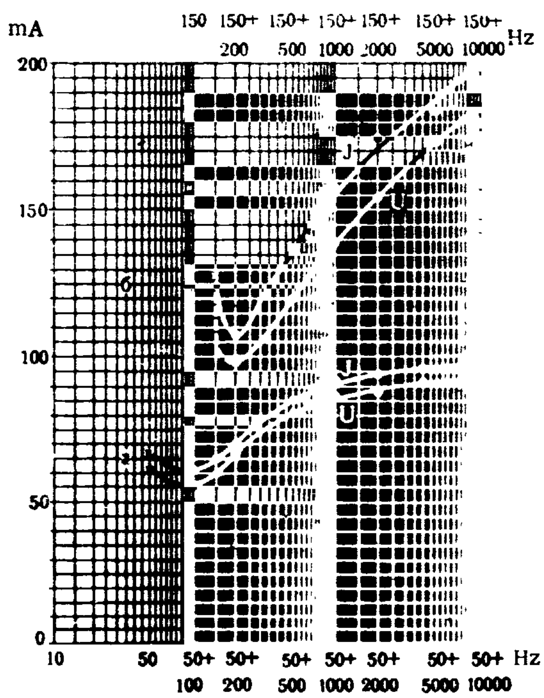

Figure 36. The Effect of Mixtures of Two Frequencies of Alternating Current on Cardiac Fibrillation (K. A. Azhibayev, I. K. Mishchenko, M. G. Turkmenov, G. L. Frenkel', V. Ya. Eskin, 1964). a, Curve of threshold-fibrillating currents in mixtures, in which basic frequency is 50 Hz (mixed frequencies are shown on the lower outline of the axis of the abscissa); b, curve of threshold-fibrillating currents in mixtures in which the basic frequency is 150 Hz (mixed frequencies are shown on the upper line of the axis of the abscissa).

Electrotrauma appearing against a background of blood loss has more severe occurrence (P. P. Goncharov, 1939; G. L. Lyuban, 1957). There are also contradictory data, however (S. F. Yakushevich and G. M. Yevel'kin, 1962); this contradiction is primarily explained by the use of different species of animals.

Important significance attaches to a fact established by I. R. Petrov (1939), concerning a decrease in resistance to electrical current among animals to whose food thyroidin is added. This fully corresponds to clinical observation (Stassen, 1936; N. K. Syronskiy and M. Ya. Saprov, 1948). Hypothyroidosis has an opposite effect (Horton, Hergt, 1936).

Exhaustion, cardiovascular diseases, tuberculosis, status thymico-limphaticus and fatigue, according to Ajello's data (1935), increased the danger of injury by electrical current. But one should bear in mind that fatigue can increase electrotraumatism as the

result of decreasing attentiveness. S. F. Libakh and I. V. Tserpinskiy (1934), /154
established that resistance to current in dogs does not change during fatigue. K. A. Azhibayev and I. K. Mishchenko were also unable to note that fatigue decreased resistance of dogs to electrotrauma.

Forms of Death From the Effect of Electrical Current. Forms of death from electrical current are significantly determined by the species of animal. Important significance attaches to the question of: is an animal capable of spontaneously emerging from a condition of cardiac fibrillation or not?

Among small animals (mice, rats, guinea pigs, rabbits, cats, and even Macacus monkeys), cardiac fibrillation has a reversible character (Lellinek, 1927; Ferris et al., 1936; I. R. Petrov, 1936; N. A. Bigdorchik, 1940; Brooks et al., 1955; Kouwenhoven, Milnor, 1955; V. Ya. Eskin, 1962, and others). At the same time, among large animals, comparable in weight to a man (dogs, sheep, pigs, calves), cardiac fibrillation does not spontaneously end (Ferris et al., 1936; I. R. Petrov, 1947; V. A. Negovskiy, 1954; Brooks et al., 1955; N. L. Gurvich, 1957; K. A. Azhibayev, 1957; I. K. Mishchenko, 1963; A. Kh. Karaseva, 1967, and others). Under the conditions of an experiment, small animals die only under the effect of a relatively high strength current with phenomena of respiratory arrest.

Notwithstanding the fact that the experimental animals significantly differ from man with respect to a number of physical and physiological signs, response reactions in them to different variants of irritation with electrical current can be near those of man.

Death from Respiratory Arrest. For certain species of animals, the respiratory form of death from electrical current is typical. During passage of a current of relatively low strength along the trunk, convulsive respiratory arrest appears as the result of tetanic contraction of the respiratory muscles. Death of the organism as the result of rigidity of the respiratory muscles can occur only during adequately prolonged passage of the electrical current through the body (Ferris et al., 1936).

During closure of the electrical current maximum expiration occurs, for the power if the expiration musculature is greater than that of the inspiration musculature (A. V. Grinberg, 1939). This significantly worsens the course of electrotrauma, for in the organism there is a great decrease in the oxygen reserve (I. K. Mishchenko, 1967). Besides this peripheral mechanism of respiratory arrest, there is also a central mechanism linked to inhibition of the respiratory center. Central respiratory arrest occurs during the action of currents of much greater magnitude than those which cause "welding" of the respiratory musculature. Certain authors consider the reason for paralysis of the respiratory center to be severe anemia of the brain occurring as the result of a decrease in arterial pressure (Urquart, 1927; Pietrusky, 1938; M. A. Khazanov, 1937).

I. R. Petrov (1947) links the respiratory disorder resulting from exposure to electrical current primarily to excitation and subsequent paresis of the respiratory center as the result of stimulation of a large number of sensitive nerve endings and interoreceptors. Weiss (cited by I. R. Petrov, 1947), sees the reason for respiratory death during electrotrauma in asphyxia which develops due to tetanic contraction of the muscles. This effect can be strengthened by spasm of the rima glottidis which appears during the passage of current along the transvulvar loop (M. T. Turkmenov, 1957), when breathing becomes impossible with yet preserved excitation of the respiratory center. In this case, tracheotomy has a lifesaving effect even with the current still running. However, this is characteristic only for transvulvar current. As A. I. /155
Grinberg (1939) showed, with the standard loops spasm of the rema glottidis is absent. With adequately high current strength or with the passage of t' current along the transvulvar loop, instantaneous paralysis of the respiratory center is possible.

I. K. Mishchenko (1966) established that the duration of lethal electrical asphyxia of the muscles is nearly 5 times shorter than the duration of lethal mechanical asphyxia. This indicates the rapid exhaustion of oxygen reserves of the organism under conditions of electrical asphyxia.

Death From Cardiac Arrest. During the passage of a current through the organism one observes various reactions with respect to the heart; these are to a significant extent determined by the strength and path of the current's passage. These reactions are extremely varied: tachycardia and bradycardia, extrasystole, blockade and, finally, the most threatening — fibrillation.

It has been established that the heart is "vulnerable" to electrical shock only in a state of its refractivity ("vulnerable period"), which on the electrocardiogram coincides with the T spike (DeBoor, 1920-1921; Andrus, Carter and Wheeler, 193[illegible]. Wegria, Moe, Wiggers, 1941; Wiggers, 1949; Ferris et al., 1936). However little is known of the fact that the vulnerable period is not uniform, but rather has two moments of increased vulnerability (Brooks et al., 1955). In the literature these two moments are called "dips"[6]. There are two dips: the first (early) has high threshold, the second (late) — low threshold (Brooks et al., 1955).

If the strength of the stimulus endured during the "dip" significantly exceeds the threshold, then here what occurs is not a uniform contraction of the ventricles, but, as a rule, slight extrasystole and even groups of them. With the subsequent increase in stimulus strength the threshold of fibrillation is reached which has the least magnitude during both "dips". With the discovery of all these facts it became clear that a significant role in the polymorphism of electrotrauma is played by a new, hitherto unknown physiological parameter

There exists a nearly unanimous opinion of the fact that ventricular fibrillation during electrotrauma in man is almost unavoidably fatal (Brooks et al., 1955; N. L. Gurvich, 1957; V. A. Negovskiy, 1960 and others). The possibility of independent cessation of ventricular fibrillation in man is extremely doubtful, although there are scattered reports on this subject from authoritative clinicians (V. Ye. Nevlin and S. Ye. Karpay, 1959; L. I. Fogel'son, 1961). But occasionally cardiac arrest is not related to fibrillation, and in these cases, apparently, it is caused by stimulation of the vagus nerve (I. R. Petrov, 1947) Finally, cardiac arrest during exposure to electrical current can occur as the result of severe stenosis of the coronary blood vessels.

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[6]"Dip" in English signifies a "drop". This term in the world literature, in a manner similar to the word "stress", has also remained untranslated into other languages

The fact that electrical current can cause coronary disorders was first indicated by Huettstrung (1934). K. A. Azhibayev (1955), who investigated the electrocardiogram during nonlethal electrotraumas in an experiment, also observed coronary changes on the EKG which indicate myocardial ischemia. K. A. Azhibayev observed such changes in tests as well, when simulating 'electroconvulsive therapy", which was confirmed by Z. A. Severova (1957), and under conditions of clinical observation.

O. I. Glazova and N. N. Syrenskiy (1939) suggests that the organic changes which are observed in the hearts of persons injured by an electrical current appear as the result of coronary spasm, i.e., are of ischemic origin. The very /156
same changes in the myocardium in patients who had received electrotrauma were observed over a period of several months by O. M. Krynskiy and L. S. Musikhin (1961). Today, in the German literature, the term "angina pectoris electrica" has even become firmly implanted, and Koeppen (1955) divides it into functional and organic.

According to the data of Ferris et al., and also according to the observations of K. A. Azhibayev and I. K. Mishchenko, myocardial ischemia during experimental electrotrauma usually vanishes rapidly. However, special investigations of K. A. Azhibayev and D. A. Alymkulov (1962) showed that these periods can also be long (up to 1 hour).

It is known that myocardial hypoxia is only capable of causing fibrillation if it is nonuniform (Brooks et al., 1955; Veek, 1960; K. A. Azhibayev and I. K. Mishchenko, 1966, and others). Uniform myocardial hypoxia, as the experiments of K. A. Azhibayev and I. K. Mishchenko (1966) showed, on the other hand, increases the threshold of the fibrillating effect of current thanks to a decrease in excitability of the myocardium.

Various pharmacological preparations alter the resistance of the heart to electrical current. Thus, the catecholamines decrease this resistance: after an injection of adrenaline fibrillation of the ventricles occurs with lesser force and lesser duration of the current effect. Acetylcholine prevents the development of fibrillation. Acetylcholine in stimulation of the vagus nerve increase the tempo of fibrillation of the auricles (Rosenblueth, 1953), and do not have any effect on the fibrillation contraction of the ventricles

(Wiggers, 1941). If atropine completely paralyzes the endings of the vagus nerve in the heart, the passage of a current is totally unreflected in the tempo of fibrillation (Urquart, 1927; I. R. Petrov, 1936, 1947).

The sympathomimetic amines increase the vulnerability of the heart to electrical current (Wiggria and Nikkerson, 1942; Hoffman, Siebens, Cranfild and Brooks, 1955). This effect has a phase character and is linked with an increased content of potassium in the blood serum. N. L. Gurvich (1957), causing fibrillation of the heart in dogs with potassium chloride, noted a decrease in the threshold of fibrillation upon the administration of adrenaline or atropine, which is linked with an increase in the cardiac tempo in response to the administration of these substances.

K. A. Azhibayev and I. K. Mishchenko (1966), in experiments performed on dogs, showed that nicotinamide leads to an increase in the threshold of electrical fibrillation of the heart, on the average 50% above the original level. /157

Electrical Shock. The shock which appears during electrotraumas belongs to the group of painful ones (I. K. Akhunbayev and G. L. Frenkel', 1960, 1967). With short term contact of man and a current-carrying object, if fibrillation does not develop and respiration does not cease, typical shock can appear with very short term and high erectile phase and with rapid transition to the torpid phase. With more prolonged passage of the current, shock appears as the result of severe pain stimulation of the receptors and nerve columns, painful convulsions of the muscles and spasm of the blood vessels (ischemic pain).

In the experiment, if the "envelope loop"[7] is not used, shock is best caused when conducting the experiment under conditions of trachial intubation and artificial respiration, for a long period of time is necessary to exhaust the nervous system.

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[7]The arrangement of the experiment must of course be such as to make it possible to exclude fibrillation of the heart. For this purpose, at the suggestion of D. A. Alymkulov, one uses a special path for passing the current through the body of the animal which N. L. Gurvich successfully termed the "envelope loop". In this arrangement all 4 electrodes are placed on the paws of the dog, an arrangement by which adequate conditions are obtained for maintaining the reflex component of the shock of the current's effect; but the force lines of the current here are maximally drawn away from the heart. A current of uniform magnitude over the "full" and "envelope" loops in the first case appears, and in the second does not cause fibrillation.

In certain cases, when passing the current along the "envelope loop", one succeeds in obtaining an entirely adequate electrical shock for human pathology, with well pronounced phaseness and extended torpid phase (Figure 37).

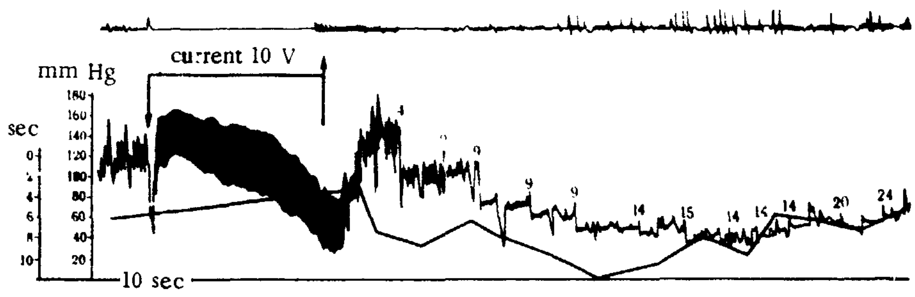

Figure 37. Electrical Shock in the Dogs With Maintenance of Spontaneous Respiration (D. A. Alymkulov, 1961). From top to bottom: respiration; arterial pressure; change in blood flow rate.

Exogenic Effects on Electrotrauma. Certain exogenic influences also alter the resistance of the organism to electrical current. Thus, overheating significantly diminishes this resistance, which is apparently caused by increased excitability of the vasomotor and respiratory centers (P. P. Goncharov, 1939). In overheated animals death frequently ensues due to primary cardiac arrest (P. P. Goncharov, 1939; A. S. Sultanaliyev, 1957).

Cooling of animals, on the other hand, increases their resistance to electrical current (V. A. Volkov, 1960). This is entirely in keeping with the fact that during the summer instances of electrotrauma increase while they decrease in the winter (Machlachan, 1934; Krakmar, 1961; V. Ya. Eskin et al., 1961).

Ultraviolet irradiation as a phase effect on the severity of electrotrauma, dependent upon the duration of irradiation: short term exposure increases the resistance of white mice to electrical current, while during more prolonged posure one observes the reverse picture (K. A. Azhibayev, 1957).

Significant interest is posed by the peculiarities of injuries caused by electrical current under conditions of altered atmospheric pressure.

A. U. Aytkulova (1957), showed that the resistance of animals (white mice) to electrical current decreases with a decrease in barometric pressure below normal.

The same data were obtained by M. D. Aksenov and A. F. Pakhomov (1953) independently of A. U. Aytkulova.

I. K. Mishchenko (1966), in investigations which were also conducted on mice, showed that a change in altitude from 760 to 3,200 meters above sea level does not alter the threshold values of voltage which causes severe in- /158
juries (3rd and 4th degree). However, during this process, transition of 3rd degree injury to 4th degree injury is observed, i.e., with a change in altitude there is an increase in the percentage of lethal injuries, which is explained by a decrease in the oxygen reserve of the organism.

All these data pertain to the respiratory form of death. With respect to the influence of altitude on cardiac activity, I. K. Mishchenko (1963), established in experiments on dogs, that the threshold of electrical fibrillation does not change during the movement of dogs from lowlands to the mountains or vice versa.

Prophylaxis and the Therapy of Electrotrauma

The prophylaxis of electrotrauma consists in safety measures, and labor hygiene which will not be examined here.

The therapy of electrotrauma depends upon the form of injury. With the respiratory form, standard methods of artificial respiration are employed (after Silvester or Schaeffer). However, to a significant extent they are less effective than intubation with rhythmic forcing of air into the lungs, for during this process the endings of the vagus nerve are stimulated and in the same way, there is adequate (rhythmic) stimulation of the respiratory center. Tsititon (cytisine solution) and lobelin (Lobeline, or inflatine), according to our observations, or are slightly effective and occasionally even worsen the condition, apparently as the result of a sharply altered (and even inverted) reactiveness of the organism which has received severe electrotrauma.

During the cardiac form, the basic task is that of eliminating fibrillation. For this purpose, short pulses of a duration of 0.01 seconds are employed with voltage on the order of 6 kV, which are obtained by the aid of defibrillation instruments. V. Ya. Eskin and A. M. Klimov created the portable defibrillator (DPA-3) with automatic current source and cardioscope, which make it possible to trace the effective supply of pulse, for defibrillation is always not eliminated with the first pulse. The intent of defibrillation consists in causing powerful extrasystole of the entire heart and thereby restoring the rhythm of the contraction.

During the shock form of injury ordinary antishock measures are carried out which are employed in the torpid phase, for in the erectile stage, as the result of its extremely rapid course, one can succeed in doing nothing. It is expedient to employ coronary dilating drugs.

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